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UPDATE ON NEUROCYSTICERCOSIS

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ABSTRACT
Taenia solium neurocysticercosis **(NCC)** is endemic in most of the world and contributes significantly to the burden of epilepsy and other neurological morbidity. Also present in developed countries because of immigration and travel, NCC is one of few diseases targeted for eradication. Neurocysticercosis is a preventable parasitic infection caused by larval cysts (enclosed sacs containing the immature stage of a parasite) of the pork tapeworm (Taenia solium). The larval cysts can infect various parts of the body causing a condition known as cysticercosis. Larval cysts in the brain cause a form of cysticercosis called neurocysticercosis which can lead to seizures. The introduction of cysticidal drugs have changed the prognosis of most patients with neurocysticercosis. These drugs have shown to reduce the burden of infection in the brain and to improve the clinical course of the disease in most patients. Further efforts should be directed to eradicate the disease through the implementation of control programs against all the interrelated steps in the life cycle of T. solium, including human carriers of the adult tapeworm, infected pigs, and eggs in the environment.

INTRODUCTION
Taeniasis and (neuro) cysticercosis are caused by the cestode Taenia solium or pork tapeworm. T. solium is a multi-host parasite with a complex zoonotic transmission cycle, circulating between the intermediate pig host and the definitive or accidental intermediate human host.[1]

T. solium infection arises from ingestion of contaminated food or water and ingestion of raw or undercooked pork and may result in taeniasis (caused by the adult tapeworm living in the small intestine) and/or cysticercosis or neurocysticercosis [NCC; caused by invasion of the larvae into the central nervous system (CNS)] in humans. Taenia solium has a complex life cycle that involves a usual intermediate host (pig) that harbors the parasitic larvae in its tissues and a sole definitive host (human) that hosts the adult tapeworm in its intestines. In the usual cycle, the adult tapeworm expels eggs or proglottids with the feces of the human definitive host, each egg containing an infective hexacanth embryo or oncosphere protected by a thick keratin embryophore.[2]

Neurocysticercosis (NCC) a major clinical consequence of T. solium infection and the dominant cause of global preventable epilepsy associated with morbidity and mortality from epileptic seizures and epilepsy related death; where T. solium is endemic, 30% of epilepsy cases are estimated to be caused by NCC.[3]

In areas with deficient sanitary conditions, free-roaming pigs have access to human feces and feed on them, ingesting the tapeworm eggs. The embryos are liberated from the eggshells and, activated by the action of gastric and intestinal juices, free themselves from the surrounding embryophoric membrane by using their three pairs of oncospheral hooks, attach to the intestinal epithelium, and actively cross the intestinal mucosa in a process facilitated by the secretion of parasite proteases. After crossing the intestinal mucosa, the embryos reach the circulatory system of the pig. Infective embryos are then distributed by the bloodstream, become established, and develop into cystic, fluid-filled larvae or cysticerci, each containing an invaginated scolex with a double crown of hooks and four muscular suckers.[4]

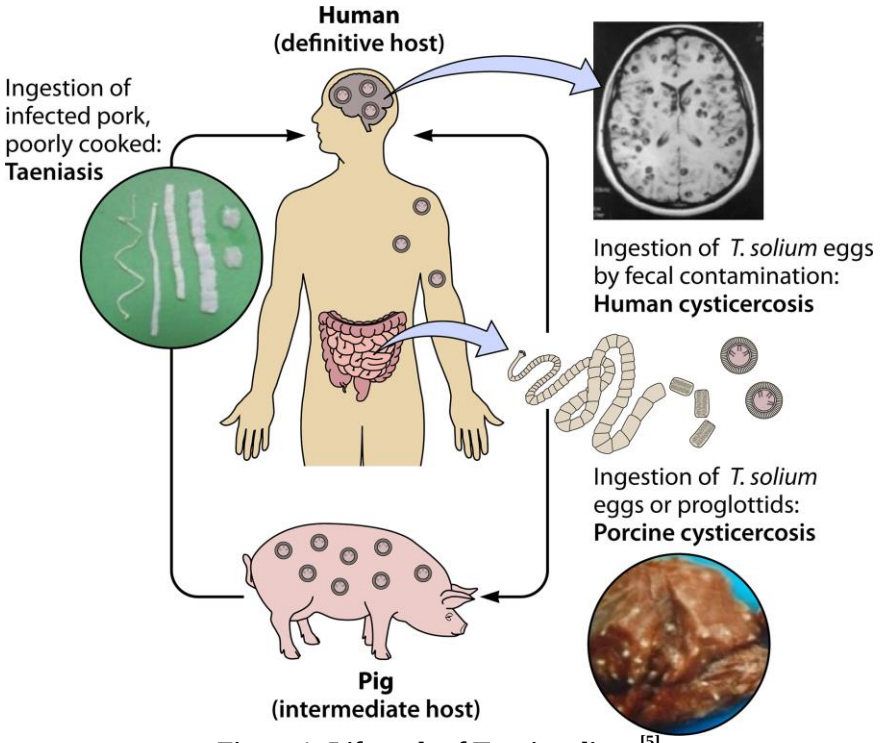

Figure 1: Life cycle of Taenia solium.[5]

Neurocysticercosis

In cases of NCC, T. solium larvae are found either in the brain tissue (parenchymal NCC) or in the intraventricular and subarachnoid spaces of the brain and spinal cord where the cerebrospinal fluid (CSF) circulates (extraparenchymal NCC) resulting in different clinical manifestations and prognoses.[6]

Extraparenchymal NCC may result in increased intracranial pressure and hydrocephalus, and patients show poorer prognosis, in part due to the growth (increase in size) of cysts in the subarachnoid space prior to symptoms becoming apparent and from late diagnosis.[7]

Types of neurocysticercosis

LOCATION	STAGE	Perilesional inflammation/edema
Parenchymal (single or multiple)	Viable Variable Degenerating Calcified	Usually present and marked
Extraparenchymal, intraventricular	Viable or in degeneration, rarely calcified	No
Extraparenchymal, subarachnoid	Viable or in degeneration, rarely calcified	Arachnoiditis or pachymeningitis, occasionally without a defined parasitic lesion

Diagnosis of Taeniasis

Serological methods enable the detection of specific anti-T. solium antibodies or T. solium antigens in the blood, urine, and CNS.

Enzyme-linked immunoelectrotransfer blot (EITB) identifies specific antibodies to lentil lectin purified glycoprotein (LLGP-EITB) antigens of T. solium.

Enzyme-linked immunosorbent assay (ELISA) detection of T. solium antibodies using crude or purified parasitic antigen extracts uses IgG as the target immunoglobulin; however, Ab- ELISAs generally have a lower specificity and sensitivity of EITB.[8]

Neuroimaging

Neuroimaging is the gold standard for NCC diagnosis. Magnetic resonance imaging (MRI) or computed tomography (CT) is used to visualize cysticerci in the CNS, providing evidence of the number of cysts, topography of lesions, stage of evolution of the cyst, and assessment of the level of the host's inflammatory reaction against parasites.[9]

Treatment of Taeniasis

Treatment options include destroying the cysts using chemotherapy, surgically removing the cysts, and/or application of symptomatic treatment (with or without removal of cysts).

Normally, therapy involves the administration of a combination of cysticidal drugs and drugs to alleviate symptoms.[10]

1) Medical Treatment

Symptomatic treatment- Patients with symptomatic cysticercosis seek medical attention because of neurological symptoms. Symptomatic medication, including analgesics, antiepileptic drugs, mannitol and steroids are in general indicated as they would be administered for seizures, headache, or intracranial hypertension from any other etiology. Symptomatic management is important and should be well established before considering the onset of antiparasitic drug therapy.[11]

Antiparasitic treatment- Praziquantel is commonly prescribed at a dosage of 50 mg/kg/day for 10–14 days; it is rapidly absorbed. Albendazole is typically given as 15 mg/kg/day for 10–14 days. In the event of severe disease and for some parenchymal cases, an extended treatment of 30 days of albendazole may be required.[12]

Complications

When a cyst is destroyed by cysticidal drugs, the resulting inflammatory reaction may be pathogenic, appearing acutely as a brain edema or chronically as a gliotic scar.[13]

Surgery

Surgery is a recommended treatment for NCC in cases of intraventricular cysts, hydrocephalus, or when the diagnosis is uncertain from neuroimaging. Calcified cysts can be removed by minimally invasive neuroendoscopy prior to the administration of cysticidal drugs as the drugs may cause the cysts to rupture.[14]

Control and elimination

Early efforts using mass human deworming with praziquantel and followed by mass chemotherapy experiences in some countries and addition of chemotherapy and vaccines to eliminate the pig reservoir increased the feasibility of interrupting transmission.[15]

Vaccination and Cysticidal Drugs for Pigs

A vaccine for use in pigs against T. solium, TSOL18 (Cysvax),has been registered for use in India since 2016 and is undergoing registration in Tanzania, Uganda, South Africa,West Africa, Kenya, Nepal, Philippines, Thailand, and Sri Lanka . Cysvax can provide 99.5% protection against porcine cysticercosis, and when combined with the anthelmintic drug, oxfendazole to deworm the pigs, protection can be increased to 99.7% effectiveness.[16]

On January 28, 2021, WHO will launch its road map for the NTDs' "Ending the neglect to attain the Sustainable Development Goals: a road map for neglected tropical diseases 2021–2030". The road map sets global targets for 2030 and includes milestones and strategies for prevention, control, elimination, and eradication of 20 diseases and disease groups and cross-cutting targets broadly aligned to the Sustainable Development Goals (SDG's). Taeniasis/cysticercosis has been targeted for control; success is defined as a steady increase in the number of countries with intensified control in hyperendemic areas, increasing from 2 (3%) in 2020 to 4 (6%) in 2023, to 9 (14%) by 2025, and to 17 (27%) by 2030. For the ambitious goals for 2030 to be met, there is a need for greater understanding of the underlying spatial epidemiology, the socio-economic drivers for pig-keeping, and social, individual, behavioral, and community perception of these neglected infections.[17]

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